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10/695,680

10/29/2003

James Frederick Harrington JR.

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EXAMINER

RAMACHANDRAN, UMAMAHESWARI

ART UNIT

PAPER NUMBER

1617

MAIL DATE

DELIVERY MODE

11/25/2008

PAPER

Please find below and/or attached an Office communication concerning this application or proceeding.

The time period for reply, if any, is set in the attached communication.

Office Action Summary**Application No.**

10/695,680

Applicant(s)HARRINGTON, JAMES
FREDERICK**Examiner**UMAMAHESWARI
RAMACHANDRAN**Art Unit**

1617

**-- The MAILING DATE of this communication appears on the cover sheet with the correspondence address --
Period for Reply**

A SHORTENED STATUTORY PERIOD FOR REPLY IS SET TO EXPIRE 3 MONTH(S) OR THIRTY (30) DAYS, WHICHEVER IS LONGER, FROM THE MAILING DATE OF THIS COMMUNICATION.

- Extensions of time may be available under the provisions of 37 CFR 1.136(a). In no event, however, may a reply be timely filed after SIX (6) MONTHS from the mailing date of this communication.
- If NO period for reply is specified above, the maximum statutory period will apply and will expire SIX (6) MONTHS from the mailing date of this communication.
- Failure to reply within the set or extended period for reply will, by statute, cause the application to become ABANDONED (35 U.S.C. § 133). Any reply received by the Office later than three months after the mailing date of this communication, even if timely filed, may reduce any earned patent term adjustment. See 37 CFR 1.704(b).

Status

- 1) ☒ Responsive to communication(s) filed on 08 August 2008.
- 2a) ☒ This action is **FINAL**. 2b) ☐ This action is non-final.
- 3) ☐ Since this application is in condition for allowance except for formal matters, prosecution as to the merits is closed in accordance with the practice under *Ex parte Quayle*, 1935 C.D. 11, 453 O.G. 213.

Disposition of Claims

- 4) ☒ Claim(s) 1-13, 17, 21 and 22 is/are pending in the application.
- 4a) Of the above claim(s) _____ is/are withdrawn from consideration.
- 5) ☐ Claim(s) _____ is/are allowed.
- 6) ☒ Claim(s) 1-13, 17, 21 and 22 is/are rejected.
- 7) ☐ Claim(s) _____ is/are objected to.
- 8) ☐ Claim(s) _____ are subject to restriction and/or election requirement.

Application Papers

- 9) ☐ The specification is objected to by the Examiner.
- 10) ☐ The drawing(s) filed on _____ is/are: a) ☐ accepted or b) ☐ objected to by the Examiner.
Applicant may not request that any objection to the drawing(s) be held in abeyance. See 37 CFR 1.85(a).
Replacement drawing sheet(s) including the correction is required if the drawing(s) is objected to. See 37 CFR 1.121(d).
- 11) ☐ The oath or declaration is objected to by the Examiner. Note the attached Office Action or form PTO-152.

Priority under 35 U.S.C. § 119

- 12) ☐ Acknowledgment is made of a claim for foreign priority under 35 U.S.C. § 119(a)-(d) or (f).
- a) ☐ All b) ☐ Some * c) ☐ None of:
- ☐ Certified copies of the priority documents have been received.
 - ☐ Certified copies of the priority documents have been received in Application No. _____.
 - ☐ Copies of the certified copies of the priority documents have been received in this National Stage application from the International Bureau (PCT Rule 17.2(a)).
- * See the attached detailed Office action for a list of the certified copies not received.

Attachment(s)

- | | |
|--|---|
| 1) <input type="checkbox"/> Notice of References Cited (PTO-892) | 4) <input type="checkbox"/> Interview Summary (PTO-413)
Paper No(s)/Mail Date. _____ |
| 2) <input type="checkbox"/> Notice of Draftsperson's Patent Drawing Review (PTO-948) | 5) <input type="checkbox"/> Notice of Informal Patent Application |
| 3) <input type="checkbox"/> Information Disclosure Statement(s) (PTO/SB/08)
Paper No(s)/Mail Date _____ | 6) <input type="checkbox"/> Other: _____ |

DETAILED ACTION

The examiner notes the receipt of the amendments and remarks received in the office on 8/8/2008. Claim 22 has been added, claims 14-16, 18-20 have been cancelled. Claims 1-13, 17, 21, 22 are pending.

Response to Remarks

Applicants' arguments regarding the rejection of claims 1-7, 12, 13, 15-17, 20, 21 under 35 U.S.C. 103(a) as being unpatentable over Harrington et al. (Spine. 2000 Apr 15;25(8):929-36) in view of Slivka et al. (US 2003/0181365, effective filing date Mar 19 2002) and further in view of Lawand et al. (Euro J of Pharmacology, 324, (1997), 169-177), rejection of claims 1, 8, 11 under 35 U.S.C. 103(a) as being unpatentable over Harrington et al. (Spine. 2000 Apr 15;25(8):929-36) in view of Slivka et al. (US 2003/0181365, effective filing date Mar 19 2002) and further in view of Lawand et al. (Euro J of Pharmacology, 324, (1997), 169-177) as applied to claims 1-7, 12, 13, 15-17, 20, 21 above and further in view of Stanfa et al. (Neuroscience, 1999, vol. 93, No. 4, p 1391-1398), rejection of claims 1, 9, 10 under 35 U.S.C. 103(a) as being unpatentable over Harrington et al. (Spine. 2000 Apr 15;25(8):929-36) in view of Slivka et al. (US 2003/0181365, effective filing date Mar 19 2002) and further in view of Lawand et al. (Euro J of Pharmacology, 324, (1997), 169-177) as applied to claims 1-7, 12, 13, 15-17, 20, 21 above and further in view of Garrett (Biol. Res. for Nursing, Vol. 1, No. 4, Apr 2000) has been fully considered and found not to be persuasive. Applicants' cancellation and addition of new claims necessitated the modified rejections presented in this action. Accordingly, the office action is made final.

Claim Rejections - 35 USC § 103

The following is a quotation of 35 U.S.C. 103(a) which forms the basis for all obviousness rejections set forth in this Office action:

(a) A patent may not be obtained though the invention is not identically disclosed or described as set forth in section 102 of this title, if the differences between the subject matter sought to be patented and the prior art are such that the subject matter as a whole would have been obvious at the time the invention was made to a person having ordinary skill in the art to which said subject matter pertains. Patentability shall not be negated by the manner in which the invention was made.

Claims 1-7, 12, 13, 17, 21, 22 are rejected under 35 U.S.C. 103(a) as being unpatentable over Harrington et al. (Spine. 2000 Apr 15;25(8):929-36) in view of Slivka et al. (US 2003/0181365, effective filing date Mar 19 2002) and further in view of Lawand et al. (Euro J of Pharmacology, 324, (1997), 169-177).

Harrington et al. teaches that disc radiculopathy can be treated with epidural glutamate receptor antagonists. The reference teaches that herniated or degenerated disc material contains free glutamate material that acts locally at the dorsal root ganglion to potentiate pain signals (p 935, key points). The reference further teaches that the injections of glutamate receptor antagonists may be beneficial in the radicular pain and other types of spinal pain (p935, lines 13-15). The reference further teaches that administration of intravenous glutamate antagonists can lessen pain responses and intrathecal delivery of glutamate antagonists can attenuate pain behavior (p 934, col. 2, lines 20-26). The reference also teaches that disc radiculopathy may be treated with epidural glutamate receptor antagonists (see Abstract). It is obvious that administration of glutamate receptor antagonists binds to the glutamate receptors and inhibits the binding of free glutamate.

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The reference does not teach a tear in a disc annulus and the administration of glutamate antagonist directly to said herniated disc tissue.

Slivka et al. teach a method of treating pain in a living being such as one caused by a bulging intervertebral disc injecting medication directly into the disc (para 044, example 2, p 7, claim 1).

It would have been obvious to one of ordinary skill in the art at the time of the invention to administer glutamate antagonist directly to said herniated disc tissue. Annulus tears can be a precursor of herniated disc or damage by tear can cause herniated disc. Harrington teaches that glutamate originating from degenerated disc may diffuse to the dorsal root ganglion and effect glutamate receptors and that local injections of glutamate receptor antagonist may be beneficial in the treatment of radicular pain and other types of spinal pain. Hence it would have been obvious to one of ordinary skill in the art at the time of the invention to administer glutamate antagonist directly to said herniated disc tissue to relieve radicular pain and spinal pain as taught by Harrington. Also, it is known in the prior art of injecting drugs or medication to the disc directly for the treatment of pain. Slivka et al. teach a method of treating pain in a living being by injection medication into the center of the intervertebral disc. Hence it would have been obvious to one of ordinary skill in the art at the time of the invention to administer medication directly to the herniated disc to alleviate pain. One of ordinary skill in the art would have been motivated in administering the glutamate receptor antagonists directly into herniated disc tissue for the treatment of pain in expectation of success because Harrington teach that local injections of glutamate receptor antagonist

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may be beneficial in the treatment of radicular pain and other types of spinal pain and Slivka teach a method of administering drugs directly into the disc to relieve pain caused by bulging intervertebral disc.

The reference does not teach an ionotropic glutamate receptor or NMDA type receptor antagonist in a method to alleviate pain in mammal.

Lawand et al. teaches the intra-articular injection in knee joint of either an NMDA or a non-NMDA glutamate receptor (CNQX) attenuated the thermal hyperalgesia and the mechanical allodynia produced by glutamate, arginine and aspartate (see Abstract). This addresses claims 2-4, 7, 12, 15, 16 and 20. The reference also teaches that the administration of MK-801 reduced the induced thermal hyperalgesic response (p 174, col. 2, lines 26-27) and thus addresses claims 5 and 6. The reference further teaches that attenuation of pain related behavior by intra-articular application of NMDA and non-NMDA excitatory amino acid antagonists after full development of the knee joint inflammation suggests a novel and viable alternative for pharmacological reduction of joint pain associated with inflammation (p 177, col. 2-7).

It would have been obvious to one of ordinary skill in the art at the time of the invention to administer ionotropic glutamate receptor or NMDA type receptor antagonist in a method to alleviate pain in mammal. The motivation to do so is taught by Harrington and Lawland et al. Harrington teach the release of free glutamate ions in disc degeneration and further teaches that local injections of glutamate receptor antagonist may be beneficial in the treatment of radicular pain. Lawland teach that intra-articular injection in knee joint of either an NMDA or a non-NMDA glutamate receptor (CNQX)

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attenuated the thermal hyperalgesia and the mechanical allodynia produced by glutamate. Hence one of ordinary skill in the art would have been motivated to administer such compounds to alleviate pain by inhibition of binding of free glutamate released (such as lumbar radioculopathy).

The references do not teach a method of alleviating pain in the elbow joint tissue of a mammal comprising administering a glutamate receptor antagonist.

It would have been obvious to one of ordinary skill in the art at the time of the invention to administer a glutamate receptor antagonist in a method to alleviate pain in the elbow joint tissue. The motivation to do so is taught by Lawland. The reference teaches that attenuation of pain related behavior by intra-articular application of NMDA and non-NMDA excitatory amino acid antagonists after full development of the knee joint inflammation suggests a novel and viable alternative for pharmacological reduction of joint pain associated with inflammation (p 177, col. 2-7). Elbow joint is another joint like knee joint and hence one of ordinary skill in the art would have been motivated to alleviate the pain in the elbow joint by administration of glutamate receptor antagonists as Lawland teaches the NMDA and non-NMDA antagonists role in attenuation of pain in knee joint inflammation.

Claims 1, 8, 11 are rejected under 35 U.S.C. 103(a) as being unpatentable over Harrington et al. (Spine. 2000 Apr 15;25(8):929-36) in view of Slivka et al. (US 2003/0181365, effective filing date Mar 19 2002) and further in view of Lawand et al. (Euro J of Pharmacology, 324, (1997), 169-177) as applied to claims 1-7, 12, 13, 15-17,

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20, 21 above and further in view of Stanfa et al. (Neuroscience, 1999, vol. 93, No. 4, p 1391-1398).

Harrington et al., Slivka et al. and Lawland et al. teachings discussed as above.

The references do not teach a method of alleviating pain by administering KA receptor antagonists and binding of free glutamate to mGlu2 receptor.

Stanfa et al. teaches the administration of non-NMDA receptor antagonists NBQX (AMPA, Glu R1-4 subunit) and LY383884, a KA receptor antagonist directly to the spinal cord of rats (col. 1, p 1392). The reference teaches the enhanced role of AMPA and Kainate antagonists in spinal nociceptive processing in inflammatory states (see Abstract) thus addressing claims 8 and 11.

It would have been obvious to one of ordinary skill in the art to use KA receptor antagonists in a method of treatment to alleviate pain. The motivation to do is provided by Harrington et al. and Stanfa et al. Stanfa et al. teaches the enhanced role of AMPA and Kainate antagonists in spinal nociceptive processing in inflammatory states. Harrington teaches the release of free glutamate ions in disc degeneration and further teaches that local injections of glutamate receptor antagonist may be beneficial in the treatment of radicular pain. Hence one of ordinary skill in the art would have been motivated to administer a KA receptor antagonist compound to alleviate pain by inhibition of binding of free glutamate released in conditions like herniated disc.

Claims 1, 9, 10 are rejected under 35 U.S.C. 103(a) as being unpatentable over Harrington et al. (Spine. 2000 Apr 15;25(8):929-36) in view of Slivka et al. (US 2003/0181365, effective filing date Mar 19 2002) and further in view of Lawand et al.

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(Euro J of Pharmacology, 324, (1997), 169-177) as applied to claims 1-7, 12, 13, 15-17, 20, 21 above and further in view of Garrett (Biol. Res. for Nursing, Vol. 1, No. 4, Apr 2000).

Harrington et al., Slivka et al. and Lawland et al. teachings discussed as above.

The references do not teach a method of alleviating pain by administering metabotropic glutamate receptor antagonists.

Garrett teaches that L-AP3 a metabotropic glutamate receptor antagonist exhibited an antinociceptive effect in animals linking effective treatment of hyperalgesia with metabotropic glutamate receptor (p 316, col. 2, lines 5-9). This addresses claims 9 and 10.

It would have been obvious to one of ordinary skill in the art to use metabotropic glutamate receptor antagonists in a method of treatment to alleviate pain. The motivation to do is provided by Harrington and Garrett. Garrett teaches the crucial role of excitatory amino acid, glutamate, NMDA and non-NMDA receptors in pain transmission, pain modulation, central sensitization and the sensation of hyperalgesia (see Abstract, p 311, col. 1, lines 15-44). The reference further teaches that L-AP3 a metabotropic glutamate receptor antagonist exhibited an antinociceptive effect in animals linking effective treatment of hyperalgesia. Harrington teaches the release of free glutamate ions in disc degeneration and further teaches that local injections of glutamate receptor antagonist may be beneficial in the treatment of radicular pain. Hence one of ordinary skill in the art would have been motivated to administer a metabotropic glutamate

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receptor antagonist in conditions like degenerated disc to alleviate pain by inhibition of binding of free glutamate released in conditions like herniated disc.

Response to Arguments

Applicants' argue that Harrington fails to describe or suggest direct administration to disc itself. In response, Harrington et al. teaches that disc radiculopathy can be treated with epidural glutamate receptor antagonists. It would have been obvious to one of ordinary skill in the art at the time of the invention to have administered glutamate receptor antagonists directly to the disc itself because Harrington teaches herniated or degenerated disc material contains free glutamate material that acts locally at the dorsal root ganglion to potentiate pain signals and further teach that local injections of glutamate receptor antagonist may be beneficial in the treatment of radicular pain and other types of spinal pain. Hence the study suggests the administration of glutamate receptor antagonist locally to alleviate the pain.

Applicants' argue that Slivka reference fails to describe or suggest the release of any free amino acids from disc material and therefore one of skill in the art would not be inclined to combine these two references. In response, Slivka has been cited to show that it is known in the prior art that medications can be injected directly to the disc and also to teach one of ordinary skill in the art the method of injecting drugs directly to disc.

Applicants' argue that Lawand does not suggest that glutamate is released from knee cartilage tissue much less disc cartilage tissue. In response, the primary reference Harrington et al. teaches the mechanism that herniated or degenerated disc material contains free glutamate material that acts locally at the dorsal root ganglion to potentiate

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pain signals and that disc radiculopathy can be treated with epidural glutamate receptor antagonists. Lawland, a secondary reference has been cited to teach an ionotropic glutamate receptor or NMDA type receptor antagonist in a method to alleviate pain in mammal. Furthermore, Lawland teaches the intra-articular injection in knee joint of either an NMDA or a non-NMDA glutamate receptor (CNQX) attenuated the thermal hyperalgesia and the mechanical allodynia.

Applicants' argue that none of the cited references describe or suggest the presence of glutamate receptors in herniated disc tissue there is no motivation to combine these references. In response one of the objectives, (Objectives, p 929, col. 1) in Harrington et al's study is "to determine whether free glutamate is available in herniated disc material in concentrations sufficient to diffuse to glutamate receptors". The reference further teaches in the results (p 929, col. 1) that densitometry of disc matrix demonstrated immunohistochemical evidence for significant extracellular glutamate and HPLC showed significant concentrations of glutamate in disc material more in herniated than in non-herniated. In conclusions (p 929, col. 1) it is stated in Harrington's study that "glutamate originating from degenerated disc proteoglycan may diffuse to the dorsal root ganglion and effect glutamate receptors. Consideration may be given to treating disc radioculopathy with epidural glutamate receptor antagonists". Hence the study suggests the presence of glutamate receptors in herniated disc as it suggests the administration of glutamate receptor antagonists for treatment of disc radioculopathy.

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Applicants' argue that rationale for combining Stanfa with Harrington, Slivka and Lawand has not been provided. In response, Harrington's teachings are directed to the release of free glutamate ions in disc degeneration and the role of glutamate receptor antagonists in alleviating pain and Stanfa et al. teaches the administration of glutamate receptor antagonists such as non-NMDA receptor antagonists NBQX (AMPA, Glu R1-4 subunit) and LY383884, a KA receptor antagonist directly to the spinal cord of rats and the enhanced role of AMPA and Kainate antagonists in spinal nociceptive processing in inflammatory states. As stated earlier it would have been obvious to one of ordinary skill in the art to use glutamate receptor antagonists such as KA receptor antagonists in a method of treatment to alleviate pain from the combined teachings of Harrington and Stanfa. One of ordinary skill in the art would have been motivated to administer a use glutamate receptor antagonist such as KA receptor antagonist compound to alleviate pain in cartilaginous tissues.

Applicants' argue that there is nothing in Garrett to support the combination of this reference with Harrington, Slivka and Lawand. In response, Harrington's teachings are directed to the release of free glutamate ions in disc degeneration and the role of glutamate receptor antagonists in alleviating pain and Garrett teaches that L-AP3 a metabotropic glutamate receptor antagonist exhibited an antinociceptive effect in animals linking effective treatment of hyperalgesia with metabotropic glutamate receptor. It would have been obvious to one of ordinary skill in the art to use metabotropic glutamate receptor antagonists in a method of treatment to alleviate pain from the teachings of Harrington and Garrett. Garrett teaches the crucial role of

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excitatory amino acid, glutamate, NMDA and non-NMDA receptors in pain transmission, pain modulation, central sensitization and the sensation of hyperalgesia and further teach that L-AP3 a metabotropic glutamate receptor antagonist exhibited an antinociceptive effect in animals linking effective treatment of hyperalgesia. One having ordinary skill in the art would have been motivated to administer a metabotropic glutamate receptor antagonist in conditions like degenerated disc to alleviate pain.

Applicants' argue that none of the cited references alone or in combination describes the foundation upon which claims are based, much less the very specific delivery route or anatomical locations required by the claims. In response, Harrington's teachings are directed to herniated or degenerated disc material that contains free glutamate material that acts locally at the dorsal root ganglion to potentiate pain signals. The reference further teaches that local injections of glutamate receptor antagonist may be beneficial in the treatment of radicular pain and other types of spinal pain. Thus the reference describes a method of alleviating pain comprising administration of glutamate receptor antagonist and suggests the local injections of glutamate receptor antagonist into herniated disc as claimed in the instant application.

Applicants' argue that without description of that foundation, there is no motivation to select the specific claimed routes of administration or anatomical locations, and the pending obviousness rejections fall short. In response, as stated above, Harrington's teachings describes a method of alleviating pain comprising administration of glutamate receptor antagonist and suggests the local injections of glutamate receptor antagonist into herniated disc. The reference further suggests

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intravenous, intrathecal deliveries of glutamate antagonists and further suggests the local injections of glutamate receptor antagonist into herniated disc. Thus Harrington et al. describes a method of alleviating pain comprising administration of glutamate receptor antagonist in degenerated disc as claimed in the instant application.

Applicants argue that without a description or suggestion that glutamate receptors exist in disc tissue, there is no reason that one of skill in the art would administer a glutamate receptor antagonist to that tissue. In response, as stated above, one of the objectives, (Objectives, p 929, col. 1) in Harrington et al's study is "to determine whether free glutamate is available in herniated disc material in concentrations sufficient to diffuse to glutamate receptors". The reference further teaches in the results (p 929, col. 1) that densitometry of disc matrix demonstrated immunohistochemical evidence for significant extracellular glutamate and HPLC showed significant concentrations of glutamate in disc material more in herniated than in non-herniated. In conclusions (p 929, col. 1) it is stated in Harrington's study that "glutamate originating from degenerated disc proteoglycan may diffuse to the dorsal root ganglion and effect glutamate receptors. Consideration may be given to treating disc radioculopathy with epidural glutamate receptor antagonists". Hence the study suggests the presence of glutamate receptors in herniated disc as it suggests the administration of glutamate receptor antagonists for treatment of disc radioculopathy.

Applicants' argue that without a description or suggestion that cartilaginous tissue of an articulating joint such as a knee or elbow is a depot/source for free glutamate, the skilled artisan would not be motivated to administer glutamate receptor

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antagonists to the joint space of such articulating joints. In response, Harrington's teachings are directed to herniated or degenerated disc material that contains free glutamate material that acts locally at the dorsal root ganglion to potentiate pain signals. The study further teaches that local injections of glutamate receptor antagonist may be beneficial in the treatment of radicular pain and other types of spinal pain. Laughlin teaches that discs are made of fibrocartilage and fibrocartilage is cartilage bundled up into dense fibers (<http://www.spineuniverse.com/displayarticle.php/article2262.html>) and it is known in the prior art that fibrocartilage is a mixture of white fibrous tissue and cartilaginous tissue in various proportions (<http://en.wikipedia.org/wiki/Fibrocartilage>). Hence from Harrington's teachings it would have been obvious to one of ordinary skill in the art at the time of the invention that degenerated disc material contains free glutamate material that acts locally at the dorsal root ganglion to potentiate pain signals and glutamate antagonists can be administered via local injections in the treatment of radicular pain. The knee and elbow are articulating surfaces or joints. It is known in the art that the menisci in the knee are made of tough cartilage and conform to the surfaces of the bones upon which they rest (<http://orthopedics.about.com/cs/meniscusinjuries1/a/meniscus.htm>) and articular cartilage is a smooth shiny material that covers the ends of the bones in the elbow (<http://jointpaininfo.com/elbow/elbowAnatomy.html>). It would have been obvious to one of ordinary skill in the art at the time of the invention from Harrington's teachings and from the prior art that herniated disc, knee and elbow joint having cartilaginous tissue. Lawland teaches the role of glutamate receptor antagonists (NMDA and non-NMDA antagonists) in attenuation of pain in knee joint inflammation. It

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is not necessary that one of ordinary skill in the art have to know all the mechanisms involved in relieving pain by administration of glutamate antagonists. It would have been obvious to one of ordinary skill in the art at the time of the invention that from Harrington's and Lawland et al.'s teachings that glutamate receptor antagonists are useful in alleviating pain in cartilaginous tissue like herniated disc, knee joints. Hence it would have been obvious to one of ordinary skill in the art at the time of the invention to administer glutamate antagonists to joint tissues like knee or elbow to alleviate pain. One having ordinary skill in the art would have been motivated to administer glutamate antagonists to degenerated disc or knee or elbow joints in expectation of therapeutic benefits such as alleviation of pain.

Conclusion

No claims are allowed.

Applicants' cancellation of claims and addition of a new claim necessitated the modified rejections presented in this action. Accordingly, THIS ACTION IS MADE FINAL. See MPEP § 706.07(a). Applicant is reminded of the extension of time policy as set forth in 37 CFR 1.136(a).

A shortened statutory period for reply to this final action is set to expire THREE MONTHS from the mailing date of this action. In the event a first reply is filed within TWO MONTHS of the mailing date of this final action and the advisory action is not mailed until after the end of the THREE-MONTH shortened statutory period, then the shortened statutory period will expire on the date the advisory action is mailed, and any extension fee pursuant to 37 CFR 1.136(a) will be calculated from the mailing date of

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the advisory action. In no event, however, will the statutory period for reply expire later than SIX MONTHS from the date of this final action.

Any inquiry concerning this communication or earlier communications from the examiner should be directed to Umamaheswari Ramachandran whose telephone number is 571-272-9926. The examiner can normally be reached on M-F 8:30 AM - 5:00 PM.

If attempts to reach the examiner by telephone are unsuccessful, the examiner's supervisor, Sreeni Padmanabhan can be reached on 571-272-0629. The fax phone number for the organization where this application or proceeding is assigned is 571-273-8300.

Information regarding the status of an application may be obtained from the Patent Application Information Retrieval (PAIR) system. Status information for published applications may be obtained from either Private PAIR or Public PAIR. Status information for unpublished applications is available through Private PAIR only. For more information about the PAIR system, see <http://pair-direct.uspto.gov>. Should you have questions on access to the Private PAIR system, contact the Electronic Business Center (EBC) at 866-217-9197 (toll-free). If you would like assistance from a USPTO Customer Service Representative or access to the automated information system, call 800-786-9199 (IN USA OR CANADA) or 571-272-1000.

/SREENI PADMANABHAN/

Supervisory Patent Examiner, Art Unit 1617

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